AMENDMENTS

In the Specification:

Please replace the paragraph on page 1, starting at line 3 with the following:

This application is a continuation-in-part of U.S. Serial No. 09/142,779, filed April 13, 1999 and which issued as U.S. Pat. No. 6,200,960 B1 on March 13, 2001, which is a 371 of PCT/AU97/00140, filed March 7, 1997, which claims priority to Australian PN 8554, filed March 7, 1996, the disclosures of each of which are incorporated herein by reference in their entirety.

A1

Please replace the paragraph on page 7, starting at line 11 with the following:

In a second aspect the present invention consists in an oligonucleotide for use in decreasing biosynthesis of Egr-1, the oligonucleotide having the sequence ACA CTT TTG TCT GCT (SEQ ID No:1).

A2

Please replace the paragraph on pages 11 and 12, starting at page 11, line 26 with the following:

In response to mechanical injury in vitro, confluent endothelial cells initiate movement into the open "wounded" area by actively responding to locally-derived signals or autocoids from injured cells. An in vitro model of vascular injury (L. Muthukrishnan, E. Warder, P.L. McNeil, *J. Cell. Physiol.* 148, 1-16 (1991)) was used to address the possible link between Egr-1 and injury-induced PDGF-B gene expression. Nuclear run-off analysis revealed that Egr-1 gene transcription was induced in cultured bovine aortic endothelial cells (BAEC) within 1 h of injury. 5' deletion analysis of the PDGF-B promoter in endothelial cells previously defined a region necessary for core promoter activity (d77) which contained a binding site for the ubiquitous transcription factor, Sp1 (L.M. Khachigian, J.W.U. Fries, M.W. Benz, D.T. Bonthron, T. Collins, *J. Biol. Chem.* 269, 22647 (1994)). Recent in vivo footprint analysis of the promoter demonstrates that the Sp1 element is indeed occupied in intact cells (R.P.H. Dirks, H.J. Jansen, B. van Gervan, C. Onnekink, H.P.J. Bloemers, *Nucleic Acids Res.* 23, 1119 (1995)). In vitro DNase I footprinting revealed that recombinant Egr-1 protected a region overlapping this site

A3

from partial DNase I digestion. When nuclear extracts from endothelial cells 1h after injury were incubated with a ^{32}P-labelled oligonucleotide spanning this region (^{32}P-Oligo B, 5'-GCTGTCTCCACCCACCTCTCGCACTCT-3') (SEQ ID No:2), a distinct nucleoprotein complex formed. The injury-induced complex was eliminated by antibodies to Egr-1. Nuclear Sp1 also bound to the PDGF-B promoter fragment; however, its levels are unaltered by injury. Thus, injury-induced endothelial Egr-1 expression precedes the induction of PDGF-B, and Egr-1 binds to a distinct region in the PDGF-B promoter also bound by Sp1.

Please replace Table 1 on page 18 with the following:

Table 1. Nucleotide Sequence of Oligonucleotides (5' –>3')

E1	CGC	CAT	TAC	CTA	GTG (SEQ ID NO: 3)
A/S2	CTT	GGC	CGC	TGC	CAT (SEQ ID NO: 4)
E6	CCA	GGC	TGG	CGG	TAG (SEQ ID NO: 5)
E7	GAG	AAC	TGA	TGT	TGG (SEQ ID NO: 6)
E9	TGT	GGT	CAG	GTG	CTC (SEQ ID NO: 7)
E11	ACA	CTT	TTG	TCT	GCT (SEQ ID NO: 8)

In the Claims:

Please amend the following claims to read as follows:

1. A method of screening for compounds which inhibit proliferation of cells selected from the group consisting of vascular cells and neoplasia cells, the method comprising determining the ability of a putative compound to inhibit induction of Egr-1, decrease expression of Egr-1 or decrease the nuclear accumulation or activity of the Egr-1 gene product.

2. The method of claim 1, wherein the method is performed *in vitro*.

3. The method of claim 1 or claim 2, wherein the vascular cells are selected from the group consisting of smooth muscle cells and endothelial cells.